American Journal of Hematology 53:251–253 (1996)

Plasma Cells Composing Plasmacytoma Have Phenotypes Different From Those of Myeloma Cells

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We describe one relapsed case of plasmacytoma of mandibular bone. The organs of relapse were liver and bone marrow. At relapse, monoclonal gammopathy (IgG-κ) was observed without suppression of IgA and IgM. By immunostaining, the plasma cells of both the original mandibular bone and liver were positive for the same cytoplasmic immunoglobulin light chain κ. The proliferative plasma cells in the bone marrow had the phenotype of $CD38^+$, $CD19^+$, and $CD56^-$ by flow cytometry and showed the presence of the rearranged IgH gene by Southern blotting. In addition, the zone of the Ig class of the patient's serum was not so sharply defined by zone electrophoresis. These results suggest that the characteristics of plasma cells of plasmacytoma are different from those of multiple myeloma. © 1996 Wiley-Liss, Inc.

Key words: plasmacytoma, multiple myeloma, rearrangement, immunohistochemistry, two-color analysis

INTRODUCTION

Plasmacytoma is one of the peripheral B-cell neoplasms recognized by the International Lymphoma Study Group. Usually, there is no monoclonal gammopathy; if present, it is low-grade. However, the majority of solitary bone plasmacytomas progress to multiple myeloma [1]. It is reported that the tumor cells of plasmacytoma are SIg^- and CIg^+, and that most B-cell-associated antigens (CD19, CD20, and CD22) are negative [2]. This phenotype appears to be similar to that of multiple myeloma. Harada et al. [3] recently revealed the phenotype of the monoclonal plasma cell (myeloma cell; $CD38^{strong\ positive\ (++)}$, $CD19^-$, and $CD56^+$), which can be distinguished from normal plasma cells ($CD38^{++}$, $CD19^+$, and $CD56^-$). Here we report on one case of solitary plasmacytoma metastasizing into the liver and bone marrow with monoclonal gammopathy, and which demonstrates the phenotypic difference from multiple myeloma.

CASE REPORT

A 65-year-old male was admitted for examination of epigastralgia. CT scan showed multiple tumors in the liver, and needle liver biopsy revealed plasmacytoid cell proliferation. Two years before admission, this case had been diagnosed as malignant lymphoma with a solitary plasmacytoma of the right mandibular bone. Bone-marrow (BM) aspirate had shown no increase of plasma cells at that time. M-protein was not detected by immunoelectrophoresis, and the levels of immunoglobulins (IgG, IgA, and IgM) were normal. After radiation therapy, the patient had been in complete remission. At admission, the analysis of peripheral blood revealed the following results: WBC count 3,500/μl, Hb levels 14.0 g/dl, and PLT count 12.5×10^4/μl. M-protein (IgG-κ) was detected by immunoelectrophoresis of serum. The levels of immunoglobulins were IgG 3,730 mg/dl, IgA 317 mg/dl, and IgM 126 mg/dl. The M-protein had increased gradually 2 months before admission. BM aspirate showed an increase of plasma cells, and the surface phenotype of these cells was $CD38^+$, $CD19^+$, $CD56^-$ (Fig. 1), $CD10^-$, $CD20^-$, IgM^-, IgD^-, κ^-, and λ^-, but the immunocytochemistry for κ was positive. The rearrangement of the IgH gene

Received for publication June 19, 1996; accepted July 10, 1996.

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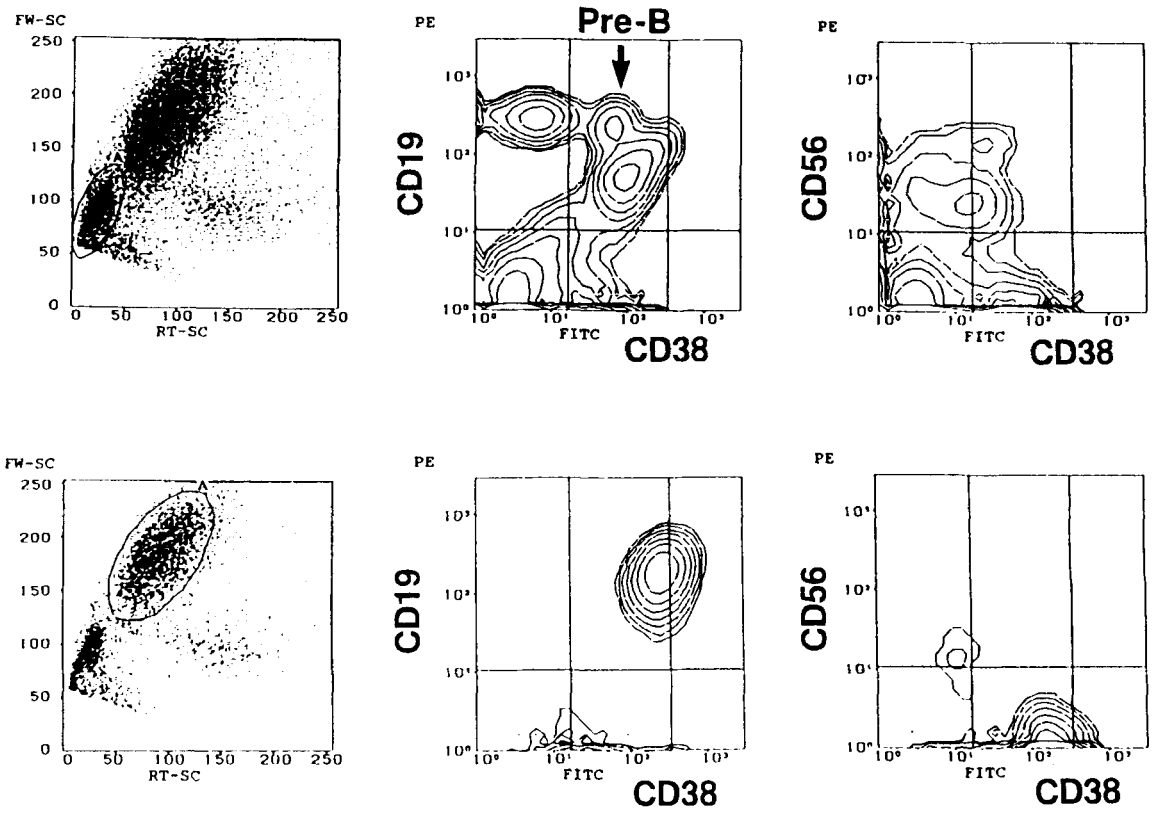

Fig. 1. Two-color analysis of BM cells with FITC-anti-CD38 antibody and PE-anti-CD19, CD56 antibody.

was detected by Southern blotting using the J_H probe. The skeletal lesions were not observed radiographically.

Immunohistochemical studies of the original right mandibular bone and the recurrent liver biopsy specimen were performed. The tumor cells were positive for both LCA (CD45) and L26 (CD20) in the bone, while positive for LCA but negative for L26 in the liver.

DISCUSSION

This case was considered a relapsed case of plasmacytoma of the right mandibular bone, because the tumor cells from the original mandibular bone, as well as liver and BM at relapse, were positive for the same immunoglobulin light chain κ. At relapse, the plasma cells increased and were shown to be monoclonal in the BM. They were negative for SIg, CD20, CD56, and CD10, but positive for CIg(κ) and CD19. The plasma cells in the liver tumor were also L26 $(CD20)^-$, κ^+ by immunohistochemistry. In serum, M-protein (IgG-κ) was observed. L26 (CD20), for which myeloma plasma cells are negative [2], was positive in the original tumor, but changed to negative in the recurrent tumors. Monoclonal gammopathy was observed. The level of immunoglobulin depends on amount of tumor; there seems to be some relationship between the expression of L26 (CD20) and the secretion of immunoglobulin.

We revealed the monoclonal plasmacytosis by Southern blotting and immunostaining in this case. However, there are several points suggesting that the characteristics of these monoclonal plasma cells were not consistent with those of multiple myeloma. First, the immunoelectrophoretic pattern of this patient's serum was different from that of multiple myeloma; the zone of the Ig class was not so sharply defined, and the normal immunoglobulins' levels were not suppressed (Fig. 2). This was supported by the observation that the considerable number of pre-B cells was found in BM by flow cytometry (Fig. 1). Tsujimoto et al. [4] reported that suppression of the pre-B cell fraction in myeloma is due to the apoptosis induced by a large number of myeloma cells. It is thought that the apoptosis of pre-B cells leads to the suppression of normal immunoglobulins in serum. Second, the tumor cells had a phenotype of $CD38^+$, $CD19^+$, and $CD56^-$. But the phenotype of myeloma cells (plasma cells) is generally $CD38^{++}$, $CD19^-$, and $CD56^+$, which could be distinguished from other hematopoietic cells in the bone marrow by two-color flow cytometric analysis [3]. Third, morphologically these cells were not so dysplastic as myeloma cells.

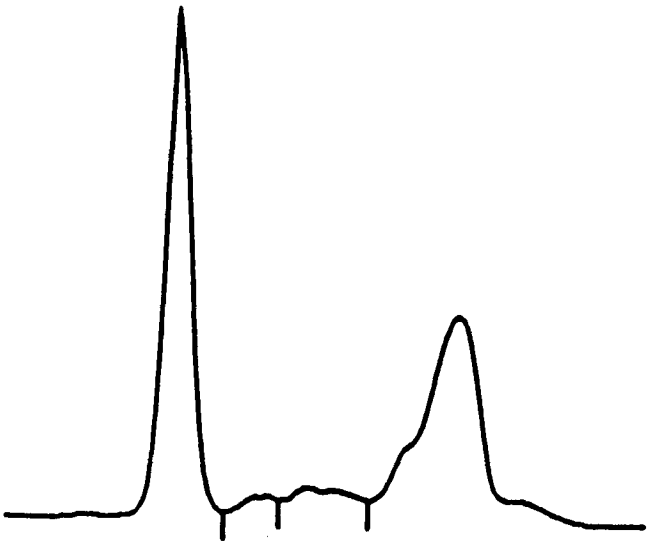

Fig. 2. Immunoelectrophoretic pattern of sera from patient.

These results suggest that plasma cells composing plasmacytoma are characteristically different from myeloma cells. In progressing to multiple myeloma from plasmacytoma, the expression of CD19 seems to disappear. The factors that regulate the expression of CD19 need to be studied.

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